

# The role of NMNAT2 in axon degeneration: relevance to Parkinson's disease.

<https://neurodegenerationresearch.eu/survey/the-role-of-nmnat2-in-axon-degeneration-relevance-to-parkinson%20s-disease/>

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## **Institution**

## **Funder**

FCT

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## **Country**

Portugal

## **Title of project/programme**

The role of NMNAT2 in axon degeneration: relevance to Parkinson's disease.

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## **The project/programme is most relevant to:**

Parkinson's disease & PD-related disorders

## **Keywords**

## **Research Abstract**

Axon pathology is a prominent feature of many neurodegenerative disorders, including Parkinson's disease (PD). Work from our group showed, in several PD cellular models, a disrupted microtubule-dependent intracellular trafficking due to reduced acetylated tubulin levels. Nicotinamide mononucleotide adenylyltransferase 2 (NMNAT2), a key cytosolic enzyme of

the NAD<sup>+</sup> salvage pathway, has been described as an endogenous axon survival factor. NMNAT2 is essential for axon sprouting, survival and maintenance during embryonic development and to preserve axonal health in the PNS. Recently, it was also demonstrated that over-expression of NMNAT2 protected against neurodegeneration in the CNS. Interestingly, our preliminary data indicates that cytosolic NMNAT2 levels are increased in PD. NMNAT2-induced NAD<sup>+</sup> replenishment could activate sirtuin-2, a NAD-dependent deacetylase, decreasing acetylated tubulin levels, which promotes axonal trafficking deficits in mature neurons. Our major goal is to investigate whether impaired NMNAT2 function, altered levels and/or cellular localization can contribute to the neurodegeneration found in PD.

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